

PULMONARY GAS EXCHANGE AND URINARY ELECTROLYTE  
EXCRETION DURING SATURATION-EXCURSION DIVING TO  
PRESSURES EQUIVALENT TO 800 and 1000 FEET OF SEAWATER

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## SUMMARY PAGE

### THE PROBLEM

To determine whether changes in pulmonary gas exchange during rest and exercise and daily urinary electrolyte excretion during saturation-excursion dives to 800 and 1000 feet of seawater (FSW) indicate beginning limitations for underwater operations at these depths.

### FINDINGS

Alveolar CO<sub>2</sub> tension, oxygen consumption, and CO<sub>2</sub> excretion did not change significantly from pre-dive control values during rest and exercise at a moderate work load (100 watts) at 800 and 1000 FSW. The respiratory pattern changes with depth. Tidal volume increased and respiratory rate decreased with increasing pressure. These findings do not indicate any respiratory limitations for the performance of moderate work at depth of 1000 FSW.

Urinary CO<sub>2</sub> excretion was significantly elevated during the saturation-excursion and decompression periods. Two subjects who complained about muscle pains in the leg, which did not respond to recompression, showed peak urinary CO<sub>2</sub> excretion either at the time the symptoms occurred or during the following days. Hyperventilation and fluid shifts due to osmotic gradients of dissolved gases have been implicated as the most likely causes of the observed pattern of urinary excretion.

### APPLICATION

This study provides additional information on the effects of a high pressure environment on respiratory gas exchange in rest and exercise. It contributes to a better understanding of different types of bends and provides support for the institution of liquid therapy during the decompression period to compensate for fluid losses occurring during the dive. The above work has significant application to the Navy Deep Submergence biomedical programs.

### ADMINISTRATIVE INFORMATION

This investigation was conducted as a part of Bureau of Medicine and Surgery Research Work Unit MR005.01.01-0063B, -- Physiological Effects of Excursion Dives from the Gas-Saturated State at Depths. The present report is No. 5 on that Work Unit. It was approved for publication on 6 March 1970 and designated as Submarine Medical Research Laboratory Report No. 615.

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## ABSTRACT

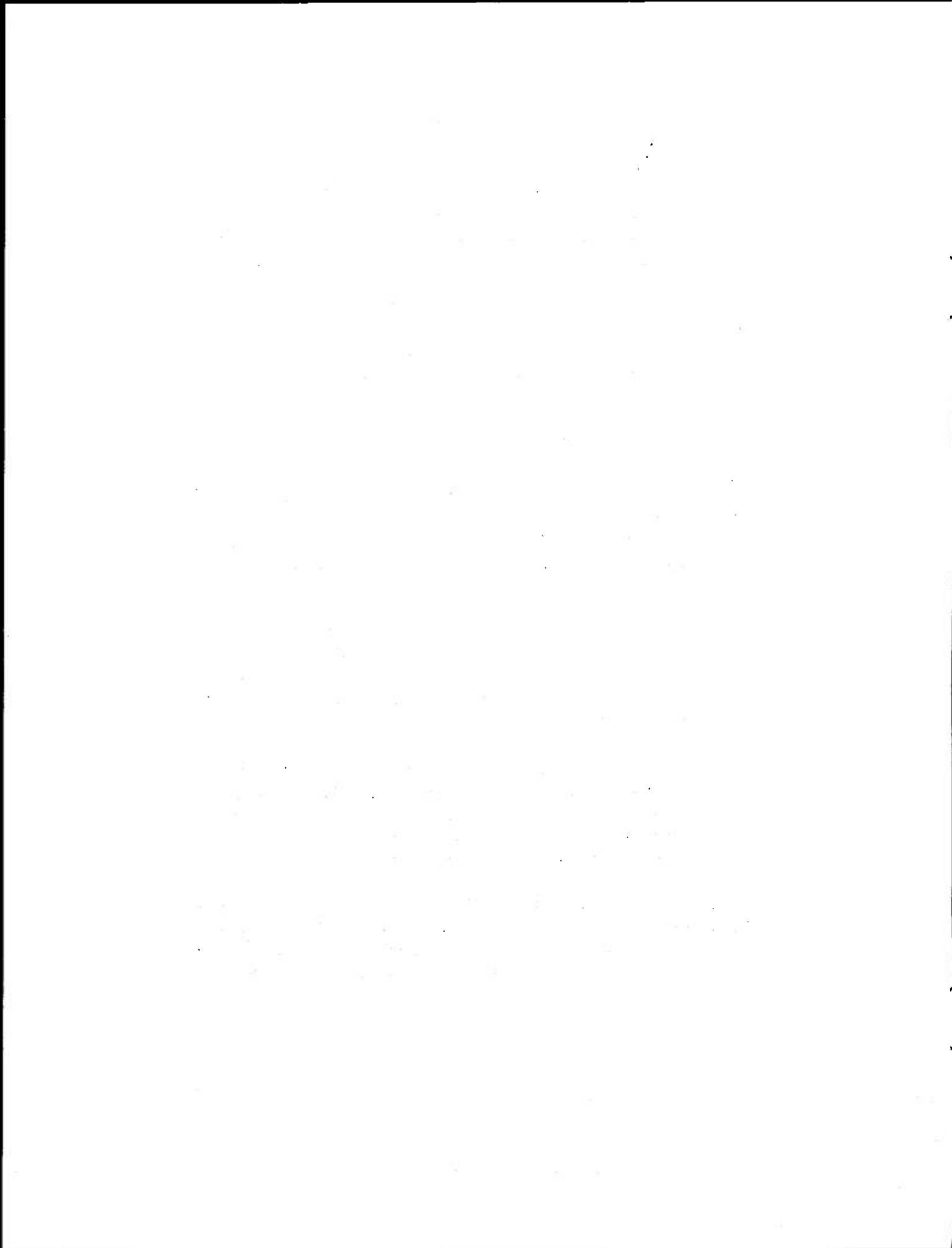
Two subjects carried out a saturation dive to 600 FSW with excursions to 800 feet of seawater (FSW). Two other subjects performed a saturation dive to 800 FSW. Both divers carried out two short excursion dives to 1050 and 1112 FSW followed later by an excursion dive to 1000 FSW lasting for 2-3/4 hours. All four divers noted helium tremors during their first excursion dive at depth exceeding 600 FSW and at very rapid compression rates ranging from 27-40 feet/min. The helium tremors were not observed during the second excursion dive at similar compression rates.

In rest and exercise,  $PA_{CO_2}$  did not change significantly at depth, but showed considerable individual variations. Pulmonary gas exchange at rest remained within normal limits at depths of 800 and 1000 FSW. Exercise performed by two subjects at a moderate work load (100 watts) at 800 and 1000 FSW resulted in an increase of  $O_2$  consumption, whereas  $CO_2$  excretion tended to decrease.

Tidal volume increased and respiratory rate decreased with increasing pressure in rest and exercise. A pronounced reduction in heart rate was observed under both conditions, which was attributed primarily to the effect of inert gas pressure, rather than to the effects of elevated  $P_{O_2}$ .

Urinary  $CO_2$  excretion was significantly elevated during the saturation-excursion and decompression periods. The two subjects who complained about muscle pains in the leg, which did not respond immediately to recompression, showed peak urinary  $CO_2$  excretion at the time the symptoms occurred.

Hyperventilation and fluid shifts due to osmotic gradients of dissolved gases have been implicated as the most likely causes of the observed pattern of urinary electrolyte excretion during the saturation-excursion period and the decompression period.



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### Introduction

Capacity to work under high pressure is mainly determined by the limitations of the respiratory system. Little is known about pulmonary gas exchange and related renal regulations of fluid and electrolyte balance during prolonged exposure to great depth. Saturation-excursion dives to pressures equivalent to 800 and 1000 feet of seawater (FSW) undertaken as a joint project among the International Underwater Contractors Inc. (IUC), of College Point, N. J., the Advanced Engineering Laboratories of Air Reduction Company (AIRCO) at Murray Hill, N. J., and the Submarine Medical Research Laboratory (SMRL), Submarine Medical Center, Groton, Conn., provided the opportunity to study lung functions, pulmonary gas exchange and urinary electrolyte excretion under high pressure.

### MATERIAL AND METHODS

The decompression schedules were developed by Mr. Andre Galerne, President of IUC. The oxygen tensions varied from 300 to 450 mm Hg. During decompression following the dives, the subjects breathed a special gas mixture (3.5 atm  $N_2$ , 1.4 atm  $O_2$ ;  $P_{O_2} = 1,000$  mm Hg), and He at different atmospheres through a respiratory mask for 10 minutes at every 100-ft level starting at 600 ft. During the last 50 ft of de-

compression, however, 100%  $O_2$  was inhaled by mask, alternating with 21%  $O_2$  in  $N_2$  every 20 minutes ( $P_{O_2} = 760$  to 1,912 mm Hg).

Our investigation involved two dives during which the subjects breathed primarily a helium-oxygen mixture with a small amount of nitrogen remaining from the air of the chamber prior to compression. The first was a saturation dive by two subjects to 600 FSW with excursions to 800 FSW. The rate of compression was two feet per minute. The divers spent a total of seven days under pressure.

The second was a saturation dive, also made by two subjects, to 800 FSW. The compression rate was 3.5 ft/min. Subject DF made a 30-minute excursion dive to 1,050 FSW, and Subject CD, a 5-minute excursion dive to 1,112 FSW. Both divers later spent 2-3/4 hours at 1,000 feet. Their total time under pressure was 13 days.

The life support system, developed by AIRCO<sup>6</sup>, during both dives provided accurate environmental control. The ambient  $CO_2$  pressure was kept under two mm Hg at all times during the dives.

Pulmonary function tests. Lung volumes and flow rates were determined by the maximal inspiratory-expiratory velocity-volume technique through the use of wedge spirometer,

a C. R. oscilloscope, and an oscilloscope camera. Results are reported in a separate communication.<sup>5</sup>

Pulmonary gas exchange. Measurements of O<sub>2</sub> consumption, CO<sub>2</sub> excretion, and alveolar gas tension were made under resting conditions and at the end of 10 minutes of exercise (100 watt work load). The divers, who were lying supine while resting samples were taken, exhaled into a Douglas bag for ten minutes. The samples of expired air, collected in evacuated steel cylinders, were later analyzed by mass spectrometry at the Naval Research Laboratory (NRL) and at AIRCO. The volumes were measured by a dry-gas meter immediately after the chamber test. Alveolar gas samples were exhaled into a rubber Haldane sample bag through a rubber tube of one meter length and two inches in diameter. The last portion of the exhaled sample was collected in evacuated steel cylinders.

The divers exercised on a Fleisch bicycle ergometer while breathing ambient gas through a low-resistance Otis-McKerrow respiratory valve. During the last two minutes of exercise, exhaled gas was collected in Douglas bags and alveolar samples were obtained in the same manner as they were under resting conditions. Samples of inspired gas were collected by opening evacuated steel cylinders near the inspiratory inlet of the respiratory valve.

In all instances, respiratory rate was recorded with a thermistor placed in the mouthpiece of the respiratory valve. Furthermore, EKG and EEG were recorded on a polygraph located

outside the chamber while the divers rested and exercised. A skullcap prepared in cooperation with Dr. Lorne Proctor's group at the Henry Ford Hospital in Detroit, Michigan, was used in obtaining the EEGs.

Twenty-four-hour urine specimens were collected in polyethylene bottles. A cover of liquid silicone (Dow Corning 200 instead of toluene) was used to prevent CO<sub>2</sub> from escaping from the urine into the chamber atmosphere. Aliquots were frozen until analyzed. Total urine CO<sub>2</sub> was determined by the manometric method of Van Slyke. Urine pH and titratable acidity were measured. Ammonia could not be determined because of insufficient quantity. A number of urine samples were analyzed immediately after collection at the Overlook Hospital, Summit, N. J., and the results agreed well with our own analyses performed later.

The subjects kept dietary log sheets and recorded the daily intake of liquid and food. Based on these notes, the daily intake was approximated. However, these figures do not include the water content of the food.

Clinical examinations and chest x-rays revealed no abnormalities in the subjects prior to and following the dive.

## RESULTS AND DISCUSSION

Vital statistics and diving experience of the subjects participating in the two saturation-excursion dives are presented in Table 1. They were healthy experienced divers, highly motivated and proud to participate in these

Table 1. Vital Statistics and Diving Experience of Subjects

Subject	Height	Weight (lbs)	Age (Year)	Previous Diving Experience
RW	6' 2"	190	25	7 years SCUBA and sport diving. Commercial diving 10 months. Bends early 1968.
BW	6' 0"	175	26	U. S. Navy diver, SCUBA, 2-1/2 of mine recovery work.
CD	5'11"	155	29	5 years experience as a SCUBA diver. No definite history of decompression sickness.
DF	5'10"	180	25	6 years experience as a SCUBA diver. Graduated from Navy under water swimmer school. Had previously some "skin bends". No definite history of decompression sickness.

studies. They expressed an interest in gaining experience under considerable higher pressures than those to which they had been previously exposed prior to actual underwater operation at greater depth. One could not ask for more cooperative subjects.

To aid the interpretation of the data obtained, a summary of the dives and a short report on the subjective experiences of the divers is given. During the first saturation excursion dive the descent to 600 FSW was accomplished within three hours and ten minutes. Short stops were made at 200 and 400 FSW for physiological and psychological tests. No symptoms were experienced during the descent. After arrival at the

"bottom", saturation depth of 600 FSW, both divers reported vague aches in joints and muscles, which were still present after a night's sleep. One of the subjects remarked that the joints in shoulder and knees felt very loose and that movements were restricted. "One has to move much more carefully than when one is on topside" (normal conditions). The excursion dive from 600 to 800 FSW was first performed by diver RW alone in a Personnel Transfer Chamber (PTC) mated to the main chamber. The descent profile was quite steep; 200 feet in 5 minutes. The diver felt a trembling which appeared to stop after a minute. He returned to saturation depth and the next day the two divers, RW and BW, went together on

an excursion dive from 600 FSW to 800 FSW in five minutes. This time only the second diver, BW, noted a trembling in the fingers. An excursion dive from 800 to 900 feet carried out by RW several hours later did not result in any symptoms. Throughout the saturation-excursion period the symptoms of "having loose joints" and dull muscle aches persisted in diver BW while the other diver, RW, who had complained about "joint stiffness" at 600 FSW prior to the excursion dive to 800 FSW did not mention such symptoms after the excursion dive. Bicycle riding with a work load of 100 watts was tolerated well by both divers. One of them, BW, noted light pain in back after the exercise, which disappeared after two minutes.

During the decompression period which lasted 100 hours 38 min. the partial pressure of oxygen was kept between 0.5 and 0.6 atm.

Diver RW reported diffuse muscle aches in legs and arms at 592 FSW after a poor night's sleep. During the next day the quality of his sleep improved considerably and the symptoms had disappeared at the time 240 FSW was reached. The other diver, BW, complained that he slept less and less well during the decompression period. He started to have intermittent pains in knees at 400 FSW, the following night he had pain in both legs which prevented him from sleeping. At 260 FSW he was placed on a face mask and inhaled a mixture of 90%-10% He-O<sub>2</sub> corresponding to a partial pressure of 0.8 atm of O<sub>2</sub>. There was a temporary slight relief of pain. Later he breathed a high nitrogen-oxygen mixture (60% N<sub>2</sub>,

20% O<sub>2</sub> and 20% He). He noted that the pain went away for a short time and then came back first in the left and then the right knee during the nitrogen treatment. At 189 feet, the intensity of the pain had increased to such an extent that a recompression to 210 feet was performed where he remained for two hours breathing a high oxygen gas mixture of 1.4 atm O<sub>2</sub> for half hour periods. This brought some relief. During the subsequent decompression period BW had intermittent pain in knees and muscles which was slightly relieved by application of hot towels. At 30 feet, another recompression treatment was instituted by bringing the chamber to 60 feet. The pain stopped for a minute, then came back and was more severe. From 50 feet on to the surface the divers alternated in breathing 100% O<sub>2</sub> and 21% O<sub>2</sub> chamber air. After reaching the surface BW had pain for about ten minutes before it started to diminish. During the following two weeks of vacation, his legs still hurt him, particularly at night, and he had sleeping difficulties. He felt he had recovered by the third week after the dive.

In the second saturation-excursion dive, the divers descended to 800 feet within 4 hours with stops at 200, 400 and 600 FSW for tests. During the compression, diver CD stood motionless for about one half hour intensely watching the depth gauge. He had placed one foot forward on a chair. After half an hour he suddenly experienced an intense pain in the buttocks which diminished somewhat during the next few hours and disappeared the next morning after a night's sleep. Both divers in the second experiment also noted the so called



looseness of joints and the restriction of movements reported by the two other divers in the first experiment. During the exercise test at 1000 FSW, CD had the curious sensation in his left ankle that the joint moved a little bit in the socket. He stopped pedalling for a moment, shook the foot a few times, had some pain, then the joint felt all right. He pedalled for another 4-5 minutes and the sensation returned. He repeated the same maneuver shaking his foot and the joint seemed to come back into a normal position.

The two excursion dives from 800 to 1100 FSW by CD and 825 to 1050 FSW by DF were carried out at compression rates of 27 and 28 FSW/min, respectively. Both subjects experienced weakness and slight tremors in both knees and thighs throughout the whole excursion dive until they returned to the saturation level of 800 FSW. One of the divers reported a strong feeling of tension in the thigh muscle and rapid tremors. He also observed a slight tremor of the hand. Another excursion dive from 800 to 1000 FSW, carried out by both divers together at a compression rate of 17 feet/min., a day later, did not produce any symptoms of tremor.

During the beginning of the decompression period at 669 FSW, CD complained about increasing pains in both knees. He related later that he had slept in a cramped position with both legs crossed. Following recompression of CD to 770 FSW, the symptoms rapidly disappeared. During the decompression period, diver DF breathed for 10 min. at several depths (600, 400, 340, and 200 FSW) a special three-gas

mixture: 3.5 atm.  $N_2$ , 1.0-1.4 atm  $O_2$  and the rest Helium. At 600 FSW, DF had no symptoms, but at each other depth the inhalation of the high nitrogen mixture produced signs of dizziness which were considered narcotic effects. The other diver, CD, had no reactions when breathing the same nitrogen mixture at 500 FSW. On change from 50 to 40 feet, both divers experienced intermittent pain in both knees which appeared again at 30 feet and was relieved by recompression to 40 feet, with the exception of numbness in the fingers of diver DF. When numbness developed also at the soles of the feet of DF, further recompression to 60 feet was carried out, which produced an improvement, but more intense pain occurred in the lower legs during the subsequent decompression at 20 feet. Another recompression to 165 feet, (Navy Diving Table 5 A), produced only partial relief. When the symptoms got worse during the following ascent phase at 60 FSW, a third massive attempt was made to clear up the symptoms by recompression up to 527 FSW without success. He stayed at this depth for about one hour having intermittent severe pain in both legs. Application of hot compresses helped to relieve the pain temporarily. After another hour, he received one Bufferin tablet at the depth of 521 feet, which in his words "acted like a hit". He slept for a while and after several hours his pain was greatly relieved and the subsequent decompression was uneventful.

The symptoms observed during the saturation-excursion dives to 800 and 1000 FSW can be classified in three categories:

1. Helium tremors occurred in all four divers during excursion dives with very high compression rates ranging between 27 to 40 feet/min. at depth exceeding 600 FSW.

2. Mechanical interference of normal circulation resulted in symptoms which occurred at two occasions and responded rapidly to recompression.

3. Aching musculature and stiffness, poorly localized, were intermittently occurring. They did not respond to recompression and oxygen treatment. As is shown later, they appear to be associated with fluid and electrolyte shifts and a large CO<sub>2</sub> excretion in the urine.

The helium tremors did not cause significant changes in psychological performance tests which is in line with observations of Brauer<sup>3</sup> and in contrast to findings of Bennett<sup>1</sup>. Performance tests, carried out by Weybrew and Parker<sup>23</sup> before and after the occurrence of helium tremors during the excursion dives from 600 and 800 FSW, showed only a slightly poorer performance in addition, letter cancelling, and form perception tests. Before and after the excursion dive from 800 to 1100 FSW of diver CD and directly during the stay of diver DF at excursion depth of 1050 FSW, choice reaction time measurements were carried out by Parker which did not show any significant differences from pre-dive control levels<sup>16</sup>. Inhalation of the high nitrogen mixture at 200 FSW by DF, which produced narcotic effects as indicated in dizziness and light-headedness, was found to be associated with a

slight decrease in the scores of the choice reaction time test. Throughout all the other phases of the saturation excursion dives of the four subjects no significant changes in the psychological performance tests were observed<sup>23, 16</sup>.

During the saturation-excursion dive to 1000 FSW, EEG recordings were obtained by Mr. van den Ende of Dr. L. Proctor's Department, Henry Ford Hospital, and C. R. Carey, SMRL. Visual inspection of the EEG record did not reveal any significant changes during the whole experiment, except a burst of slow waves of about eight seconds duration in CD at 800 FSW. This abnormality, diagnosed as an electrical storm, did not recur during the subsequent exercise test or during the following part of the experiment. Computer analysis of the records did show some minor frequency changes related to compression, saturation and decompression, which will be reported in a separate communication.

In summary, it can be stated that the saturation-excursion dives to 800 and 1000 FSW produced subjective symptoms, but the effects on the CNS were apparently so small that they did not cause any marked changes in the EEG or a significant impairment of performance, as indicated in the results of psychological tests.

Pulmonary gas exchange. Data on respiratory minute volume (BTPS), respiratory rate, tidal volume, pulse rate, PA<sub>CO<sub>2</sub></sub>, CO<sub>2</sub> excretion, O<sub>2</sub> consumption, and respiratory exchange ratio obtained on all four subjects at 800 FSW and during decompression at 400 FSW are presented, together with

pre-dive and post-dive surface values, in Table 2. Respiratory minute volume increased under pressure based on an increase in tidal volume.

The four subjects'  $PA_{CO_2}$ 's varied considerably at depth. Each of the two divers participating in the saturation-excursion dive from 600 to 800 FSW exhibited one peak in  $PA_{CO_2}$ , one of 57 and the other of 64 mm Hg (see Fig. 5). Otherwise,  $PA_{CO_2}$  remained within the normal range. Although we took alveolar samples at more frequent intervals in the 1000-ft dive, we obtained no evidence of elevated  $PA_{CO_2}$  in the two subjects; to the contrary, the  $PA_{CO_2}$  tended to decrease both during rest and exercise. If taken at comparable periods at depths and lumped together, data on the four subjects revealed no significant change in  $PA_{CO_2}$ . Oxygen consumption increased slightly more than  $CO_2$  excretion did at depth under resting conditions. The first two subjects, who showed higher  $PA_{CO_2}$  at depth, also exhibited larger  $CO_2$  excretion at depth than did the other two subjects.

Table 3 shows similar data collected on two subjects during moderate exercise (100 watt) breathing  $HeO_2$  at depths of 800 FSW and 1000 FSW, as well as pre- and post-dive control values breathing air. In addition, data were collected during moderate exercise nine months later, breathing air and a mixture of 60%  $O_2$  - 40%  $N_2$ . Respiratory minute volume was amazingly constant under all conditions,

whereas tidal volume increased and respiratory rate declined under pressure. Breathing 60%  $O_2$  - 40%  $N_2$  resulted in a very slight increase in tidal volume and small decreases in respiratory and pulse rates. Although the changes were in the same direction, their magnitude was much larger under pressure than when the subjects breathed 60%  $O_2$  - 40%  $N_2$ . The  $PA_{CO_2}$  did not change significantly, but tended to decrease as it did when the subjects rested. Excretion of  $CO_2$  decreased slightly when the subjects exercised, whereas  $O_2$  consumption increased, causing the respiratory exchange ratio to decrease.

The change we observed in breathing patterns at greater depth--an increase in tidal volume, and a decrease in respiratory frequency at rest and during exercise--is consistent with the findings of earlier investigators (8, 10, 18, 20). This change is probably caused by breathing gases of high density, rather than by the increased  $P_{O_2}$  at depth.

This conclusion is supported by comparing the changes occurring in tidal volume and respiratory frequency during exercise at 1000 FSW with an inspired  $P_{O_2}$  at 450 mm Hg with the

changes occurring under the same exercise load at one atm abs at a corresponding inspired  $P_{O_2}$  of 459 mm Hg.

At depth, tidal volume increased 23%; at sea-level pressure, 8%. Respiratory frequency decreased 25% and 2.2%, respectively.

The 100-watt work load with a minute ventilation of 30 liters imposed no ventilatory restrictions at 1000 FSW

Table 2. Effect of Exposure to High Pressure on Pulmonary Ventilation and Gas Exchange in Resting Conditions. (Means and Standard Errors for four subjects)

Condition	$\dot{V}_E$ BTPS (liters/ min)	Resp. Rate (breaths/ min)	Tidal Volume (liters)	Pulse Rate (beats/ min)	$PA_{CO_2}$ (mm Hg)	$\dot{V}_{CO_2}$ (liters/ min)	$\dot{V}_{O_2}$ (liters/ min)	RQ
Control predive	6.88 .60	11.4 1.56	.617 .041	80.3 5.0	36.5 1.03	.225 .015	.254 .011	.88 .04
Compression at 800 FSW	8.27 .62	11.1 1.3	.788 .132	64.8* 3.0	35.9 2.1	.289 .044	.371 .068	.78 .01
Decompression at 400 FSW	9.26* .44	9.8 .9	.961* .069	60.3* 4.5	32.9 1.8	.225 .098	.285 .074	.79 .03
Surface postdive	8.62 1.02	10.6 1.7	.858 .167	76.3 5.1	37.9 2.6	.289 .042	.278 .028	.89 .02

\* Statistically significant difference from control, at the 5% level and better.

Table 3. Effect of Exposure to High Pressure on Pulmonary Ventilation and Gas Exchange during Exercise (100 watts, Mean for two subjects)

Condition	$\dot{V}_E$ BTPS (liters/ min)	Resp. Rate (breaths/ min)	Tidal Volume (liters)	Pulse Rate (beats/ min)	PA <sub>CO<sub>2</sub></sub> (mm Hg)	$\dot{V}_{CO_2}$ (liters/ min)	$\dot{V}_{O_2}$ (liters/ min)	RQ
Control predive on air	33.6	23.4	1.407	135.6	35.7	1.205	1.306	.93
800 feet on He-O <sub>2</sub>	30.05	19.2	1.567	101.8	34.4	1.085	1.406	.77
1000 feet on He-O <sub>2</sub>	30.3	17.5	1.720	94.8	33.2	1.030	1.446	.78
Immediate postdive on air	33.6	22.7	1.476	115.8	33.5	1.078	1.272	.85
9 months postdive on air	31.6	23.0	1.360	126.5	37.5	1.146	1.169	.89
9 months postdive on 60% O <sub>2</sub> -40%N <sub>2</sub>	31.6	21.8	1.470	124.6	36.8	1.093	1.250	.87

when HeO<sub>2</sub> was breathed. Consequently, there was no evidence of CO<sub>2</sub> retention when environmental CO<sub>2</sub> was below measurable concentrations (1 to 2 ppm)--observations that stand in contrast to our previous findings that PA<sub>CO<sub>2</sub></sub> increases during prolonged exposure to a He-O<sub>2</sub>-N<sub>2</sub> atmosphere at 200-FSW pressure with an equivalent sea level CO<sub>2</sub> content of 1.1% (20). The importance of maintaining adequate environmental control in high-pressure experiments is obvious.

The rather large individual variations both in PA<sub>CO<sub>2</sub></sub> and CO<sub>2</sub> excretion that we observed in our four subjects (all trained divers) under high pressure have also been reported by other investigators<sup>2</sup>. However, the differences observed in the subjects of the first and second dives cannot be related to their training and diving experience which has been previously shown to influence the alveolar ventilation in response to an increase in airway resistance. 13, 14, 15, 18, 20, 21

Since the minute ventilation showed no response to the 100 watt work load at depth while the O<sub>2</sub> consumption increased, the ventilation equivalent for O<sub>2</sub> ( $V_E/V_{O_2}$ ) decreased, suggesting an improved O<sub>2</sub> transfer at depth from the atmosphere to the blood. The elevated PI<sub>O<sub>2</sub></sub> at depth could not have caused this decrease, since the ventilation equivalent did not change markedly when the same exercise was performed at a corresponding higher PI<sub>O<sub>2</sub></sub> at one atm abs pressure. Similar reductions in the ventilatory equivalent of O<sub>2</sub> under

pressure have been reported by Hamilton<sup>7</sup>.

There was a pronounced reduction in heart rate at rest, and exercise under pressure. Similar findings have been previously reported by Hamilton in a helium-oxygen saturation dive at 620 feet<sup>7</sup>. This decrease in heart rate could be produced by two factors: (1) increased partial pressure or oxygen (300 - 450 mm Hg) and (2) the increased gas density. For the exercise tests we are able to differentiate the contributions of the two factors, since the tests were repeated with the same work load at normal atmospheric pressure with a PI<sub>O<sub>2</sub></sub> of 459 mm Hg corresponding to that at 1000 feet (450 mm Hg). The decrease in pulse rate due to increased oxygen tension at normal atmospheric pressure amounted to 1.5%, as compared with a 30% fall in pulse rate at 1000-foot depth. This is in line with observations of Hesser, et al<sup>9</sup>, who found that only part of the reduction in heart rate observed during exercise at 4.5 atm breathing air was due to the effects of the increased inspired oxygen tension and the major part was related to the effect of increased gas density.

As a result of the reduction in heart rate at depth, there is an increased oxygen pulse (ratio of oxygen consumption per minute to pulse rate) during exercise at depth which increased from 9.6 prior to the dive to 15.3 at 1000-foot depth. There is only a slight increase from 9.2 to 10.0 during exercise at an elevated PI<sub>O<sub>2</sub></sub> at normal atmospheric pressure.

The increase in oxygen pulse at depth, which helps to maintain the oxygen supply to the tissues at a lower rate, appears to be part of a regulation known to exist in subjects having an increased parasympathetic activity as is frequently the case in well-trained athletes.

It appears likely that not only the bradycardia at depth, but also the respiratory pattern of increased tidal volume and decreased frequency, are the results of a vagal stimulation produced by exposure to high pressure.

Urinary excretion. Urinary excretion data are shown in Tables 4 and 5. Urinary volume tended to increase both during the saturation-excursion dives and the decompression periods, a notation which is in agreement with previous observations<sup>20,22</sup>. Urinary pH, CO<sub>2</sub>, and bicarbonate increased significantly during exposure to high pressure; titratable acidity changed little, and chloride excretion decreased. The average calcium excretion did not change, while organic phosphorus and sodium excretion increased significantly during the saturation-excursion period. Average daily excretion of urine electrolytes are shown in Figs. 1, 2, 3. Unfortunately, ammonia excretion could not be determined, because of insufficient samples; no estimate of net hydrogen ion excretion could, therefore, be made.

The average daily liquid intake of the four divers during the control period was 1.84 liters. There was an increase during the first day of the saturation-excursion period, associated with the exposure to high temperatures

due to compression. During the second day of the saturation-excursion period and during the subsequent decompression period liquid intake decreased, reaching the lowest value at the last day of decompression (Fig. 4).

Daily changes in urinary CO<sub>2</sub> excretion and  $P_{ACO_2}$  observed in the 800-FSW dive are shown in Fig. 5. The  $P_{ACO_2}$  of both divers remained within a normal range, with the exception of one peak noted for each diver following compression to 600 and 800 FSW. Urinary CO<sub>2</sub> excretion increased only slightly in one diver, but greatly in the other. The latter developed pains in both knees and legs at 180 FSW during decompression. The pain was not relieved by a two-hour recompression to 210 FSW during which O<sub>2</sub> at 1.4 atm was breathed through a face mask for 30-minute periods. Pain persisted for two weeks after the experiment.

Similar urinary measurements were made during the second dive to 1000 FSW (Fig. 6). The  $P_{ACO_2}$  tended to decrease slightly both during the saturation-excursion dive and decompression. Urinary CO<sub>2</sub> excretion of both subjects was elevated throughout the exposure. Subject DF exhibited a high peak on the day that he suffered severe pain in both knees and legs at 50 FSW during decompression.

These two cases of bends-like symptoms developed, of course, during decompression, when at 10-minute intervals the divers breathed O<sub>2</sub> by mask at 1000 mm Hg in N<sub>2</sub>. It appears, therefore, that symptoms consisting of muscle pain and vague stiffness that do

Table 4. Effect of Exposure to High Pressure on Urinary Electrolyte Excretion  
(Means and Standard Errors for four subjects)

Conditions	Fluid Intake L/24 hrs	Urine Volume L/24 hrs	Urinary pH	Total CO <sub>2</sub> mEq/24 hrs	HCO <sub>3</sub> mEq/24 hrs	Titratable Acidity mEq/24 hrs
1. 2-day control period prior to dives on air	1.84 .21	.930 .125	6.480 .06	1.18 .25	1.08 .31	37.8 5.2
2. 3-day period saturation- excursion dives 2 subj: 600-800 FSW 2 subj: 800-1000 FSW	1.575 .26	1.260 .097	6.695* .059	3.54* .98	2.92* .46	34.2 5.1
3. 4-day period decom- pression	1.148* .188	1.173 .090	6.65* .03	4.71* .98	3.52* .77	30.2 3.5

\* Differences from controls statistically significant at the 5% level and better.



Table 5. Effect of Exposure to High Pressure on Urinary Electrolyte Excretion  
(Means and Standard Errors, Four Subjects)

Condition	Sodium mEq/24 hrs.	Potassium mEq/24 hrs.	Chloride mEq/24 hrs.	P O <sub>4</sub> mEq/24 hrs.	Calcium mEq/24 hrs.
1. 2-day control period prior to dive on air	116.8 4.1	60.5 10.9	278.0 14.5	1.03 .09	.113 .015
2. 2-day period saturation- excursion dives 2 <u>S</u> : 600-800 FSW 2 <u>S</u> : 800-1000 FSW	141.8* 10.2	62.95 6.68	223.5* 18.1	1.44* .13	.112 .016
3. 4-day period decom- pression	126.0 8.3	50.13 4.4	241.6 17.0	.96 .09	.127 .013

\* Differences from controls statistically significant at the 5% level and better.

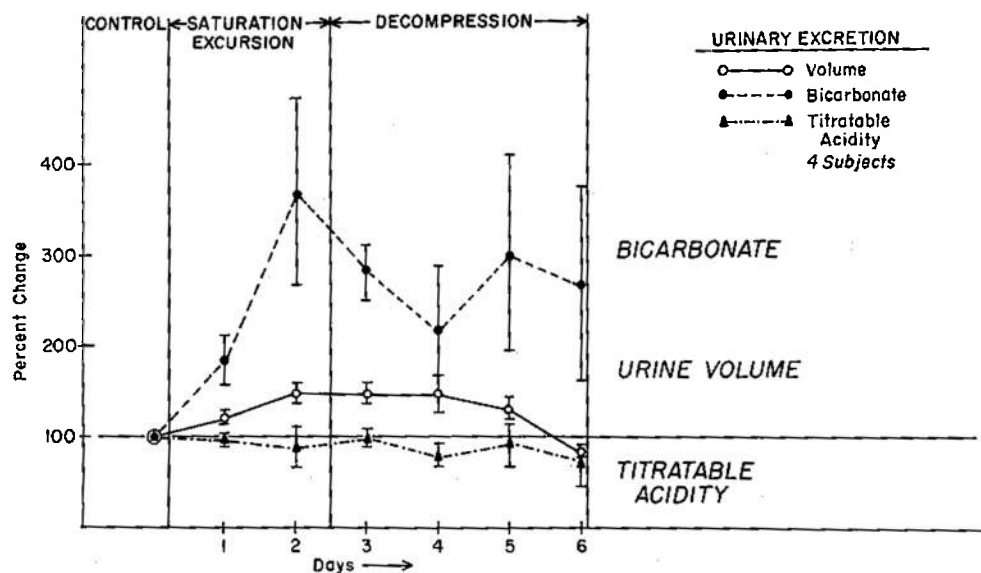


Fig. 1. Daily changes in urine volume, urinary bicarbonate and titratable acidity excretion of four subjects during saturation-excursion dives to 800 and 1000 FSW and during four days of decompression.

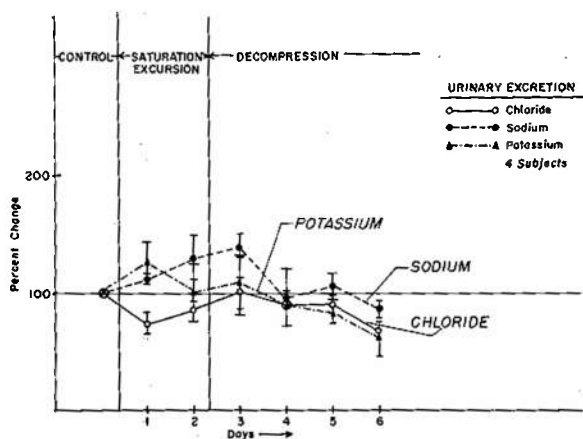


Fig. 2. Daily changes in urinary chloride, sodium and potassium excretion of four subjects during saturation-excursion dives to 800 and 1000 FSW.

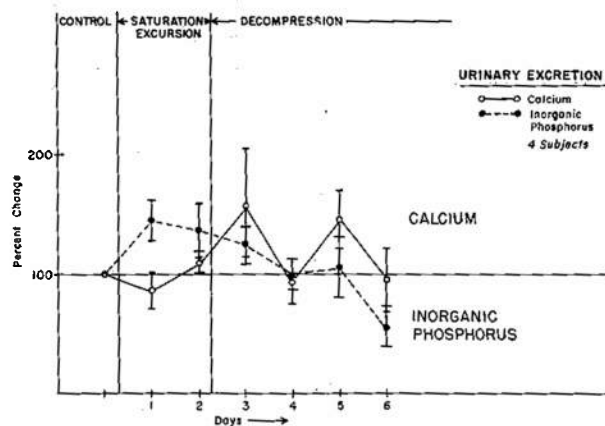


Fig. 3. Daily changes in urinary calcium and phosphorus excretion of four subjects during saturation-excursion dives to 800 and 1000 FSW.

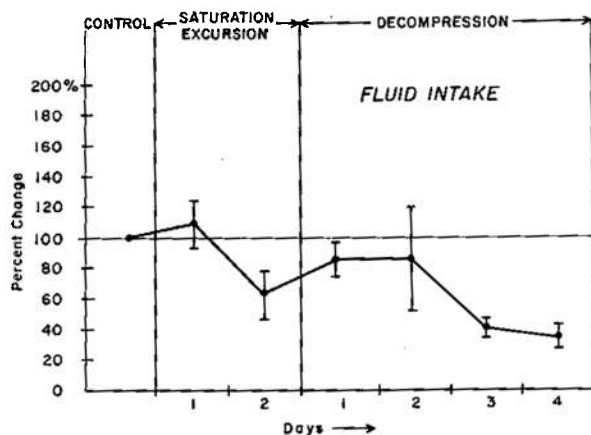


Fig. 4. Daily changes in liquid intake of four subjects during saturation-excursion dives to 800 and 1000 FSW.

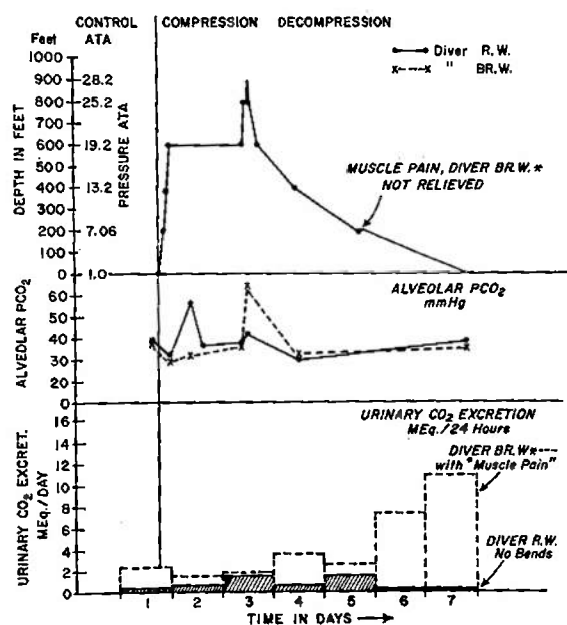


Fig. 5. Diving profile, showing alveolar  $CO_2$  tension and urinary  $CO_2$  excretion of two subjects during saturation dive to 600 FSW with an excursion dive at 800 FSW.

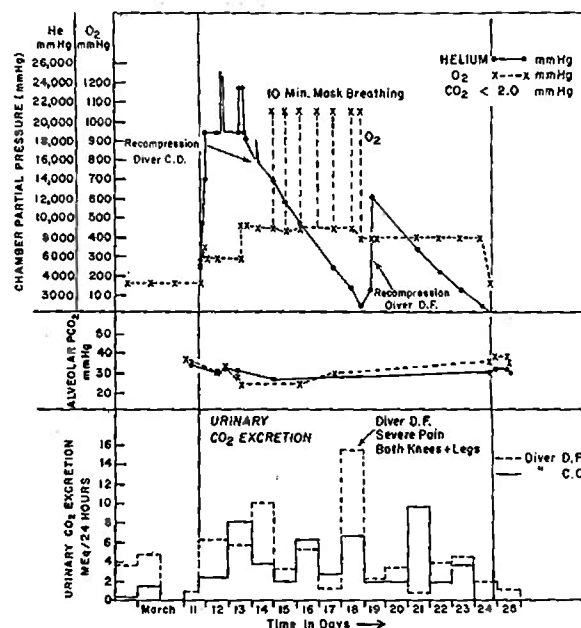


Fig. 6. Diving profile, showing partial pressure of He and  $O_2$ , alveolar  $CO_2$  tension, and urinary  $CO_2$  excretion of two subjects during saturation-excursion dive to 1000 FSW (34.3 atm abs.) decompression and recompression of divers.

not seem to respond to recompression are related to the combined effects of high pressure of He and increased  $P_{O_2}$ , as reported by Bennett<sup>1</sup>.

Since the titratable acidity declined rather than increased during saturation and decompression, it appears that renal compensation of an increased acid or  $CO_2$  load is not needed. This assumption is supported by the fact that the average alveolar  $CO_2$  tension of the four divers remained normal and decreased slightly in the two subjects participating in the second experiment. The hypothesis advanced by Kylstra et al<sup>11</sup> that osmotic gradients are produced by dissolved gases, therefore, appears pertinent. If during rapid compression the partial pressure of inert gases in the blood exceeds the partial pressure in the poorly perfused tissues, osmotic gas gradients would cause water shifts from poorly perfused tissues to the blood and better perfused tissues. The extracellular volume might thereby increase, causing the more profuse urinary excretion that we observed in our subjects during the saturation-excursion period and during three days of decompression period.

The observations on liquid intake support this assumption. Only during the first day of the saturation-excursion period was the slightly elevated urinary volume associated with an increased liquid intake. Throughout the rest of the experiment the volume was increased in spite of a decreased liquid intake.

Since increased bicarbonate excretion is associated with decreased chloride excretion, it appears that the

former acts to conserve chloride. This mechanism is a defense of the chloride component of the plasma under conditions of fluid volume shifts. The excessive amount of bicarbonate excreted during decompression by the divers whose muscle pains did not respond to recompression suggests that greater fluid shifts contributed to the development of their symptoms. It is pertinent to compare the pattern of electrolyte excretion observed in these saturation-excursion dives to 800 and 1000 FSW in which ambient  $CO_2$  was below measurable levels with previously reported findings in a saturation dive at 200 FSW pressure with an equivalent  $CO_2$  content of 1.1%<sup>20,21</sup>. In the latter experiment, alveolar and mixed expired  $CO_2$  was found to be significantly elevated and the urinary electrolyte excretion exhibited a response characteristic of hypercapnia. Urine volume,  $CO_2$  and titratable acidity were markedly elevated. Potassium, sodium and chloride excretion showed a transient increase lasting for four days. In contrast to these previous findings, we observed in the present experiments a decrease in titratable acidity together with a significant rise in pH and a decrease in chloride excretion which appear to reflect a more alkalotic state. Under conditions of respiratory alkalosis due to hyperventilation, chloride is retained and bicarbonate excretion is increased<sup>17</sup>. The tendency to decreasing alveolar  $CO_2$  concentration in the two divers of the second experiment and their post-dive blood data which show an increase in serum chloride and a decrease in serum bicarbonate as compared with their predive blood values (Table 6) seem to indicate that hyperventilation played a

Table 6. Haematological and Blood Electrolyte Values (A) Prior to and  
(B) After Saturation-Excursion Dive to 1000 FSW

	Subjects			
	DF		CD	
	A	B	A	B
RBC mill/cmm	4.6	4.82	4.6	4.97
Hgb. g/100 ml	15.1	15.3	15.4	15.0
Hct.	41	44	46	43
Total Leucocytes	7400	11000	9500	12800
Total Neutrophils	4366	5940	6650	9216
Total Lymphocytes	2072	4180	2470	2432
Na mEq/L Plasma	145	144	142	141
K mEq/L Plasma	5.3	5.1	4.8	4.4
CL mEq/L Plasma	102	106	96	106
CO <sub>2</sub> mEq/L Plasma	27	25	29	27

role in the observed pattern in urine electrolyte excretion. The post-dive values for leucocyte were markedly increased, which has also been observed in earlier investigations<sup>20,22</sup>. The importance of hyperventilation in saturation-excursion dives has been emphasized by Brauer<sup>3</sup> in his report on his 1189 FSW dive. Bühlmann, who has carried out a considerable number of dives to great depths mentions that he carefully instructs his subjects not to hyperventilate during the dive. He attaches great significance to the prevention of hyperventilation<sup>4</sup>.

The observed reciprocal changes in increased bicarbonate excretion and decreased chloride excretion indicate that conditions existed during the saturation-excursion dives which favored chloride reabsorption instead of bicarbonate. Such conditions exist both in hyperventilation and in fluid shifts a circumstance resulting in an increased extracellular volume and increased urinary excretion. Increased blood volume is known to inhibit aldosterone secretion leading to a decreased absorption of sodium and decreased excretion of hydrogen ions and increased water loss. The reduction in sodium reabsorption may result in a decrease of bicarbonate reabsorption; while chloride reabsorption shows opposite changes to bicarbonate. Hyperventilation and fluid shifts resulting in an increased extracellular volume could explain, therefore, the increase in urine volume, bicarbonate and sodium excretion, and the decrease in titratable acidity, hydrogen ion, and chloride excretion observed during the saturation-excursion dives. At the end of the decompression period, the fluid

shifts should be reversed since the osmotic gradients caused by the dissolved gas would favor an influx of fluid from the better perfused tissues to the poorly perfused tissues. If this is so, one should expect a decrease in urine volume and a decrease in sodium excretion and an increase in chloride excretion conditions which correspond with the data obtained during the decompression period as shown in Tables 4 and 5 and Figures 1 and 2. The sustained loss of fluid occurring during the saturation-excursion period and the first part of the decompression period and the reduction of fluid intake during the decompression period must result in a decrease of circulating blood volume towards the end of the decompression period when the fluid shifts towards the poorly perfused tissues with high residual gas content are presumably at their peak. This, in turn, would contribute to a reduction of gas exchange and bubble resorption and result in the occurrence of bends. The events of the second saturation-excursion experiment to 1000 FSW are pertinent. Both divers experienced intermittent pain during the last part of the ascent at 50 FSW (5th day of decompression). One responded to recompression treatment, the other did not. The latter reported that he sweated all the time throughout the whole experiment and that changes in humidity did not produce an improvement. It is therefore most likely that DF had a markedly greater fluid loss than CD, a condition which caused a greater interference with gas exchange and bubble resolution. His reported symptoms of numbness and tingling sensation in the feet suggest that he hyperventilated and it is safe to assume that he continued to

hyperventilate during the long hours of uncertainty associated with the three recompression attempts and intervening decompression periods. Such a long-lasting hyperventilation period could be responsible for the peak bicarbonate excretion during this day. The extraordinary analgesic effect of one tablet of Bufferin should be evaluated against the background of a state of hyperexcitability in which the subject experienced intense pain and a throbbing pulse.

The results of our experiments have a bearing on decompression treatment. The two incidents of bends which did not respond to (1) increase in total pressure, (2) increase in partial pressure of oxygen, and (3) increase in partial pressure of nitrogen, measures designed to speed resolution of gas bubbles, were associated with high urinary bicarbonate excretion. The latter has been implicated as an indicator of electrolyte imbalance due to hyperventilation and fluid shifts. It therefore seems indicated to follow the suggestion of Lambertsen<sup>12</sup> to possibly institute a form of fluid therapy during the decompression period following saturation-excursion dives. This might simply consist of drinking 2.0 liters of water per day during the decompression period to overcome the observed tendency towards a reduction in fluid intake and to compensate for the fluid losses occurring during the dive.

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13. ABSTRACT Pulmonary gas exchange and related renal regulation as indicated in urinary electrolyte exchange were measured during saturation-excursion dives to 800 and 1000 feet of sea water (FSW) in four subjects for the purpose of determining beginning physiological limitations of underwater operations at these depths. Alveolar CO <sub>2</sub> tension, oxygen consumption, and CO <sub>2</sub> excretion did not change significantly from pre-dive control values during rest and exercise (100 watt) at 800 and 1000 FSW, which indicates that there is no respiratory limitation for moderate work load at the depth of 1000 FSW. Urinary electrolyte excretion showed a characteristic pattern during the saturation-excursion period, consisting in increased urine volume, large rise in bicarbonate excretion, and decrease in chloride excretion. Hyperventilation and fluid shift due to osmotic gradients of dissolved gases have been implemented as the most likely cause of the observed pattern of urinary excretion. Bends which did not respond to recompression and were found to be associated with large increases in bicarbonate excretion appear to be caused by excessive fluid shifts. It is suggested that in such cases liquid therapy should be instituted to compensate for the loss of liquid during the dive.		

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